ORIGINAL ARTICLE

Postictal Aphasia and Paresis: A Clinical and Intracerebral EEG Study

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ABSTRACT: ***Background:*** We examined the lateralizing value of postictal language and motor deficits and studied their underlying mechanisms. ***Patients and methods:*** The total sample consisted of 35 patients (26 temporals, 8 frontals, 1 parietal) with a good postsurgical outcome (Engel's class I and II). Postictal examination was blindly reviewed on videotapes. In 15 cases (29 seizures), postictal language manifestations were analyzed in relation with the diffusion of the epileptic discharge recorded by intracerebral EEG. Language dominance was determined by the intracarotid amobarbital test. ***Results:*** Postictal aphasia was observed only when (1) seizure originated in the dominant hemisphere and (2) ictal activity spread to language areas (Wernicke and/or Broca areas). When the epileptic focus was in the nondominant hemisphere, no postictal aphasia was observed even if there was secondary generalization of ictal activity affecting the language areas of the dominant hemisphere. Postictal motor deficits also had a strong lateralizing value even when seizures were secondarily generalized. ***Conclusion:*** Postictal aphasia in temporal epilepsies and postictal motor deficits in temporal and extra temporal epilepsies provided excellent lateralizing information. Postictal deficits appear to be the result of inhibitory mechanisms induced by previous ictal activity of the structures related to these functions.

RÉSUMÉ: Aphasie post-ictale et parésie: étude clinique et électroencéphalographique intracérébrale. ***Introduction:*** Nous avons examiné la valeur de latéralisation post-ictale des déficits du langage et des déficits moteurs et nous avons étudié les mécanismes sous-jacents. Patients et ***Méthodes:*** L'échantillon comprenait 35 patients ayant subi avec succès une chirurgie (classe I et II de Engel), dont 26 ayant présenté un ictus temporal, 8 un ictus frontal et 1 un ictus pariétal. L'examen post-ictal a été révisé à l'aveugle sur bande vidéo. Dans 15 cas (29 crises), les manifestations post-ictales au niveau du langage ont été analysées en relation avec la diffusion de la décharge épileptique enregistrée par ÉEG intracérébral. La dominance pour le langage a été déterminée par le test intracarotidien à l'amobarbital. ***Résultats:*** L'aphasie post-ictale a été observée seulement quand 1) les crises originaient dans l'hémisphère dominant et 2) l'activité ictale s'étendait aux zones du langage (zones de Wernicke et/ou de Broca). Quand le foyer épileptique était situé dans l'hémisphère non dominant on n'a pas observé d'aphasie post-ictale, même quand il y avait généralisation secondaire de l'activité épileptique affectant les zones du langage de l'hémisphère dominant. Les déficits moteurs post-ictaux avaient également une grande valeur de latéralisation, même quand les crises étaient secondairement généralisées. Conclusion: L'aphasie post-ictale dans les épilepsies temporales et les déficits moteurs post-ictaux dans les épilepsies temporales et extratemporales fournissent de l'information précieuse sur la latéralisation. Les déficits post-ictaux semblent être le résultat de mécanismes inhibiteurs induits par une activité ictale antérieure des structures reliées à ces fonctions.

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Analysis of the clinical characteristics of spontaneous seizures constitutes a fundamental aspect of the presurgical evaluation of patients with partial epilepsies. Although a number of studies have shown the high lateralizing value of ictal and postictal verbal manifestations,[1-11] some[12] have questioned its relevance when there is secondary generalisation of seizure activity. In addition, while ictal speech manifestations do occur in extra temporal seizures,[6] the lateralizing value of postictal aphasia might be limited to temporal lobe cases.[6,10] The mechanisms underlying the appearance of postictal aphasia remain unclear[9,13] although, in accordance with cortical stimulation data,[14-19] a direct relation to ictal activity, and its intracerebral diffusion, has been postulated. The literature on the lateralizing value of postictal motor function is more limited and, since the reports of Todd, Gowers and Jackson[20-23] who observed postictal paresis on the side most affected by the seizure, no significant series of cases have been published. The neuronal

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explanation of postictal motor deficits has been debated in terms of cellular exhaustion[20,22-23] or inhibitory processes,[21,24] this latter possibility coming from the observation of postictal paralysis without preceding motor activity during seizure.

In order to study the lateralizing value of postictal aphasia and paresis and their underlying mechanisms, a large series of temporal and extra temporal epileptic patients was examined for language and motor functions during EEG-video recordings. Postictal language manifestations were analyzed as a function of ictal discharge origin and diffusion as recorded by intracerebral EEG while postictal motor deficit was examined as a function of ictal motor signs.

Patients and Methods

Patient population

Thirty-five patients (18 men, 17 women) followed at Notre-Dame Hospital for severe and medically intractable seizures were included on the basis of: 1) their excellent postsurgical outcome (class I or II of Engel[25] after a mean follow-up of 8 years [5 - 15 years]), and 2) availability of postictal language and motor assessment during EEG-video recordings. All patients were investigated presurgically according to a standardized protocol[26] that relies on clinical, neuropsychological, imaging data (computed tomography scan and magnetic resonance imaging), and surface and intracerebral video-EEG recordings of ictal and interictal activities. Invasive EEG recordings were performed in 32 patients with stereotaxically implanted electrodes according to a lateromesial approach. Three to six electrodes were implanted in the frontal and temporal lobes according to where the seizures possibly originated.[27] Additional electrodes could be inserted in other locations, determined on an individual basis.

Twenty-six patients had temporal lobe epilepsy and nine had extra temporal lobe epilepsy (eight frontals and one parietal). Sixteen temporal cases had seizure onset in mesial temporal structures, seven simultaneously in mesial and lateral temporal cortex and two in lateral temporal cortex. Seizures of the seven frontal patients investigated with depth-electrodes originated from supplementary motor area (SMA; one case), anterior cingular gyrus (one case) or more diffuse unilateral frontal areas (five cases). The parietal patient had a right precuneus focus. Five temporal lobe patients had a second, less active contralateral focus. However, seizures analyzed in this study all originated from the main focus. Fifteen patients were operated on the left (13 temporals, two frontals) and twenty on the right side (13 temporals, 6 frontals, 1 parietal). Neuropathological data were available for 27 patients and showed mesiotemporal sclerosis in 11 temporal cases, space-occupying lesion in three temporal and four extra temporal cases, posttraumatic scar or gliosis in two extra temporal cases and no abnormality in five temporal and one frontal cases. Mean age at surgery was 27 years (range: 12 - 43 years).

The cerebral dominance for language was confirmed by the intracarotid amobarbital procedure (IAP) in all cases, except in three right-handed frontal lobe epilepsy patients. However, according to the literature,[14] these patients have a high probability of left hemisphere language dominance (92 to 99%).

Thirty-one patients were tested for postictal language manifestations (24 temporals, six frontals, one parietal) and 30 for postictal motor function (22 temporals, eight frontals).

Analysis of postictal language and motor functions

Videotape recordings of language and motor postictal testing were reviewed retrospectively by one of the authors (Ch. A.) who was unaware of the focus lateralization and localization, and to the spread of the discharge. Equivocal manifestations were subsequently reviewed by a second blinded observer (I.R.) to clarify the interpretation. Most of the seizures examined were complex partial; some were simple partial or secondarily generalized. Language assessment was usually performed with a nonstandardized protocol which included naming (objects and body parts), execution of simple verbal commands, counting and reading. In some cases, a modified version of the Montréal-Toulouse protocol[28] was administered. It consisted of picture naming, designation (word-picture matching), and repetition of words and simple sentences.

One hundred and forty-two (142) seizures assessed for postictal language deficits were included in the analysis (mean: 5, range: 2-10 seizures per patient). We excluded seizures for which only spontaneous speech was available since some patients may correctly utter some words and simple sentences while still being aphasic when more specific tests are used.

Postictal motor function was usually evaluated by asking patients to extend their arms in front of them. Sometimes patients were asked to close their eyes and to show their teeth. Testing was performed by the EEG technician, as soon as possible after the end of the ictal phase. Seventy-eight seizures (78) were evaluated for motor function (mean: 3, range: 1 - 4 seizures per patient).

The determination of the postictal phase onset was based on clinical observations (arrest of automatisms or tonic-clonic manifestations, reaction to the observers, return of a normal behavior). This determination was usually easy; and if not obvious on the videotape, the end of the seizure was verified on EEG recordings. A brief period of postictal confusion could follow the end of a seizure. In this case, language was assessed after confusion had disappeared.

Postictal aphasia and intracerebral seizure diffusion

In 15 patients (13 temporal, two extra temporal), the relationship between seizure diffusion and postictal language manifestations was studied through the examination of intracerebral recordings. In these patients, two electrodes were particularly relevant: one temporal and one frontal. The temporal electrode exploring the posterior hippocampus and the posterior part of the middle temporal gyrus was, on the dominant hemisphere, close to Wernicke's area.[17-19] When this electrode was absent (three cases), activity recorded from the lateral contact of midtemporal electrode was examined. The frontal electrode exploring the anterior cingular gyrus was situated, on the lateral convexity of the dominant hemisphere, just in front of Broca's area.[16] Two patients did not have frontal electrodes. For the purpose of our analysis, language areas were considered affected by the seizure if intracerebral activity involved the whole dominant hemisphere or, at least, the lateral contacts of the two electrodes described above. When the right hemisphere was dominant for language (two temporal cases), these electrodes were used to examine the diffusion of the ictal

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discharge to language areas (on the right hemisphere) even though we were aware that the intrahemispheric organization of language functions might not be a mirror-image of what is observed when the left hemisphere is dominant for language.

Statistical analyses

The statistical analyses were performed with Student's t-test or Fisher's exact test.

RESULTS

Language postictal testing

Among the 31 patients who were tested for postictal aphasia, 24 patients (77% of cases) were easily classified as either aphasic (12 patients) or non-aphasic (12 patients).

All aphasic patients presented with both receptive and expressive abnormalities except two patients who showed severe expressive deficits such as long-lasting anomia and numerous paraphasias without clear receptive anomalies. All aphasic patients showed postictal aphasia after most of their seizures (56/59, 95% of the seizures). Three patients who were usually aphasic after their seizure, presented one seizure without postictal aphasia (either a simple partial seizure (n=2) or a complex partial seizure (n=1)). This emphasizes the importance of carefully assessing language functions after more than one seizure per patient. Among these 12 patients, 9 had their epileptic focus in the dominant hemisphere and 3 had a bilateral speech representation according to the result of the amobarbital test. All these patients had a temporal lobe focus.

Twelve patients were non-aphasic. No language abnormalities were observed in these cases, not even speech arrest in the early postictal period. All the 12 non-aphasic patients had their epileptic focus in the non-dominant hemisphere. Nine of these patients had a temporal lobe focus, two had a frontal lobe focus and one had a parietal lobe focus.

The remaining seven patients (23% of patients, 26 seizures) were classified as uncertain concerning the presence of postictal aphasia. Only mild perturbations were observed, often isolated to one aspect of language. Failure rates were generally low, many patients making only one or two errors. In addition, the majority of the errors observed were not truly aphasic, i.e., perseveration, unrelated verbal paraphasia, failure to respond, English answer in a bilingual francophone. These deficits appeared intermittently (absent in 50% of seizures). In two patients who received an extensive language evaluation, the mean duration of language disturbances was much shorter than in clearly aphasic patients (1 min. 15 sec. versus 4 min. 9 sec.). This pattern was more frequent in frontal lobe (4/6, 66%) than in temporal lobe patients (3/24, 12.5%, $p < .05$). Among these seven patients, only one frontal had their focus in the dominant hemisphere.

Postictal motor function

Among the 30 patients tested for postictal motor function, 12 (40% of cases) presented a lateralized partial deficit, that is, eight temporal (36% of temporals, 19 seizures) and four frontal lobe patients (50% of frontals, eight seizures). The motor deficit involved the face (three temporals, one frontal), the upper limb (two temporals, one frontal), or both (three temporals, two frontals), and was always contralateral to the epileptic focus ($p<0.01$). This unilateral deficit occurred in five patients even when the seizure was secondarily generalized (all four frontals and one temporal, seven seizures). Motor tonic, clonic or tonic-clonic ictal signs preceded postictal deficit in 10 cases and were maximal on the side of the deficit. Two other patients (with a temporal lobe focus) presented with a postictal motor deficit despite no tonic or clonic motor signs during the seizure (seven seizures).

Postictal language functions and intracerebral seizure diffusion

The relationship between the presence of postictal aphasia and intracerebral propagation of seizure activity was examined in 15 patients (29 seizures) all with unilateral dominance for language. The results of this analysis are presented in Table 1. There were six patients in the aphasic group (all temporals) and nine patients in the non-aphasic group (seven temporals, one frontal, one parietal).

In the aphasic group, 13 seizures were analyzed. In 11 seizures followed by postictal aphasia, intracerebral ictal discharge widely propagated to the whole dominant hemisphere involving Wernicke's area (10 seizures) and/or Broca's area

Table 1: Relationship between postictal aphasia and intracerebral seizure diffusion

A) Seizures originating from *dominant* hemisphere

Case	Number of seizures	Postictal aphasia	Diffusion to W and/or B	SEEG ictal onset
1	1	+	+	L mes. T
2	1	-	-	L mes. T
	1	+	+	L mes. T
3	1	+	+	L mes. + lat. T
	1	+	+	L mes. + lat. T
4	2	+	+	R mes. + lat. T
5	1	+	+	L mes. T
	1	+	+	L mes. T
6	1	-	-	L mes. T
	3	+	+	L mes. T

B) Seizures originating from *nondominant* hemisphere

Case	Number of seizures	Postictal aphasia	Diffusion to W and/or B	SEEG ictal onset
7	1	-	+	R mes + lat. T
8	2	-	+	R lat. T
9	1	-	-	R mes. + lat. T
10	1	-	-	R mes. + T
11	1	-	-	R precuneus
12	3	-	-	R mes. T
13	3	-	+	L mes. + lat. T
14	2	-	+	R mes. T
15	2	-	+	R F

Abbreviations: **W**: Wernicke's area, **B**: Broca's area, **L**: left, **R**: right, **mes**.: mesial, **lat**.: lateral, **T**: temporal, **F**: frontal

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(seven seizures). In two patients (no. 3 and 9), who were usually aphasic but had one seizure that was not followed by postictal aphasia, the ictal activity remained localized to the amygdala and the hippocampus on the dominant hemisphere.

In the non-aphasic group, 16 seizures were examined. In six seizures, Broca's and Wernicke's areas were not affected by the seizure. In 10 other seizures (62%), ictal activity widely propagated to the dominant hemisphere (contralateral to the epileptic focus), involving Wernicke's and/or Broca's areas.

Thus, in our sample of patients with unilateral language representation, postictal aphasia occurs only when 1) the seizure focus is in the dominant hemisphere *and* 2) the seizure activity propagates to language areas. When the seizure focus is located in the nondominant hemisphere, postictal aphasia does not occur even if seizure activity propagates to language areas. These results apply mainly to temporal lobe epilepsy since only two extra temporal cases were included in this analysis.

Discussion

The present study confirms the value of assessing postictal language and motor functions in determining the lateralization of the epileptic focus. Most relevant clinically is the finding that postictal manifestations have a lateralizing value even when the seizure is secondarily generalized contrary to what has been claimed previously by some authors.[12]

Despite some confusion in properly separating the postictal from the ictal period, and some limitations in the availability of postictal observations, recent studies have confirmed the value of postictal aphasia in lateralizing the seizure focus. This is especially true when EEG-video monitoring of postictal manifestations is available, and when language dominance is confirmed with IAP.[6-9] However, the usefulness of postictal aphasia to correctly lateralize the focus varies greatly from study to study (from 49% to 100%), depending both on the method used to assess language functions and on the characteristics of the sample (i.e., whether only temporal lobe epileptics are included). Moreover, it must be emphasized that postictal language testing should be performed after each patient's seizure. Erroneous conclusions (absence of postictal aphasia) may be reached when ictal activity originating from the dominant hemisphere does not spread to the speech areas. This occurs mainly in simple partial seizures.

Our study does not support the hypothesis that difficulties in interpreting postictal language manifestations are due to confusion as reported by Theodore et al[29] and Devinsky et al.[9] Indeed, even if confusion was encountered in many patients, it disappeared early and was followed either by normal speech or by aphasia that lasted much longer (a mean of more than four minutes). This point of view is shared by Helmstaedter et al[30] in the context of another cognitive postictal paradigm evaluating verbal and nonverbal memory.

The results that were obtained with frontal lobe patients are more difficult to interpret. In these patients, postictal language perturbations were usually mild, short-lasting, and not truly aphasic (perseverations, unrelated verbal paraphasias, etc). Some word finding deficits were also noted. These manifestations were not related to confusion. Thus, rather than a true postictal aphasia, these manifestations can be viewed as reflecting a frontal lobe semiology, already present in our frontal lobe patients but possibly accentuated in the postictal period. This hypothesis could be verified by including, in postictal assessment protocols, tasks that are sensitive to frontal lobe dysfunction, both verbal and nonverbal. Since only few extra-temporal cases with a dominant hemisphere focus were included in our sample, it is not possible to evaluate the hypothesis that postictal aphasia does not occur after extra temporal seizures.[6,10]

The sensitivity of postictal motor examination (40%) was lower than the sensitivity of postictal language testing although it was much higher than that reported in other studies (3,5%).[4] Usually the deficit is mild and brief and tends to be more frequent in frontal lobe (50% of cases) than in temporal lobe epilepsies (36% of cases). The relatively high frequency of motor deficits in our temporal cases has not been reported before. This might be because EEG-video monitoring is relatively recent and allows a better analysis of postictal manifestations that are usually partial and brief, and intermingled with other preeminent signs. Motor deficits were initially described by Bravais[31] in 1827, then by Todd[20] in 1854, who gave his name to this paralysis. In general, the postictal motor disorders involve the limb(s) most affected by epileptic seizure.[22-23] Although Bergen et al[32] described two cases of bilateral postictal paralysis after seizures originating from the mesiofrontal cortex (likely the supplementary motor area (SMA)), our data suggest that postictal motor deficit is generally unilateral and can be considered as a reliable lateralizing sign easy to observe, even in temporal lobe cases.

Thus, language and motor postictal examination provide two good lateralizing markers of epileptic foci and their combination increases the probability of lateralizing the foci to 89% (compared to 40% for motor and 77% for language assessment separately). Similar findings were reported recently by Williamson et al.[33]

Besides its clinical relevance, the originality of this study relies on the evaluation of the postictal deficit in relation to the diffusion of seizure on intracerebral EEG recordings allowing some insight regarding their underlying mechanisms.[9,13] Postictal language disturbances were related both to the lateralization of the focus in the dominant hemisphere and to the spread of ictal activity to the language areas. Indeed, in our sample, two patients demonstrated a clear postictal aphasia when seizures originating from the dominant hemisphere temporal lobe propagated to language areas but showed no language disturbances when the ictal activity remained confined to mesiotemporal structures of the same hemisphere. These facts have been already reported in two patients studied by Privitera et al[8] and are consistent with the results of the data of the physiological studies based on electrical stimulations of brain structures if one is allowed to draw a parallel between effects of ictal activity and electrical stimulus.[14-19]

Although necessary, the ictal involvement of speech areas is not sufficient to produce postictal aphasia since seizures arising from the nondominant hemisphere and spreading to language areas were never followed by aphasia in our sample (62% of the seizures of non-aphasics). This observation is in agreement with Devinsky et al[9] who found no relationship between the degree of language alteration following right temporal lobe seizures and the amount of propagation of ictal activity to the contralateral dominant hemisphere.

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The fact that motor dysfunctions always occurred on the side contralateral to the epileptic focus despite the secondary generalization of tonic-clonic manifestations (in five patients and seven seizures) might follow the same rule, i.e., ictal activity in the vicinity of the focus does not disturb the functions the same way distal propagation of ictal activity does. In fact, some studies have demonstrated that postictal slow EEG abnormalities[34] and peri-ictal SPECT modifications of perfusion[35] tend to be predominant on the side of the epileptic focus even when there was a bilateralization of seizure activity. It is possible that the more direct propagation of intrahemispheric than inter-hemispheric ictal discharges (as assessed intracranially by the degree of coherence of ictal activity[36-37]) might explain their different functional consequences.

Our data do not support the theory of neuronal exhaustion introduced by Todd[20] and supported by Jackson.[22-23] Although motor deficits are generally observed in the limbs first and most affected by seizure, the presence of postictal paresis despite no motor signs during seizures (seven seizures in two of our temporal cases), an observation already made by Gowers[21] and Erfron,[24] does not support this hypothesis. Instead, this fact suggests the existence of some inhibitory mechanisms much more widespread than is the actual ictal activity. These inhibitory mechanisms would be particularly efficient on the side of the origin of the ictal discharge. The structures involved in postictal motor disturbances could be either cortical or subcortical; SPECT studies have shown involvement of basal ganglia in temporal lobe seizures with motor components.[38]

In summary, the postictal clinical state depends mainly on the lateralization of seizure onset: a postictal deficit results from the involvement of a functional area on the side of the epileptic focus. On the contrary, the involvement of functional areas by an ictal discharge originating in the contralateral hemisphere does not produce a postictal deficit. Postictal examination of language provides reliable lateralizing information in temporal lobe seizures and postictal motor deficit is as valuable in temporal as in extra temporal cases.

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